offset of T). QTDex was the longest minus the shortest exercise QT. Exercise perfusion images were divided into 5 segments with each segment scored for the intensity of tracer (0–3 scale). Scores for the 5 segments were added to produce an ischemia score. Coronary disease extent was the number of vessels with ≥70% stenosis.

Results: Those with ischemia scores >0 had higher mean QTDex than those with ischemia scores = 0 (45 ± 2.8 vs 33 ± 2.8 msec; $p < 0.004$). Those with and without multivessel disease had mean QTDex that did not differ (45 ± 7.3 vs 41 ± 2.3 msec; $p = 0.79$). Both QTDex ($p < 0.006$) and coronary disease extent ($p < 0.001$) were independent predictors of ischemia score. However, while ischemia score was a predictor ($p < 0.004$) of the extent of coronary disease, QTDex was not ($p = 0.23$).

Conclusion: Although both QTDex and the extent of coronary disease correlate independently with the extent of ischemia, QTDex was not an independent predictor of the extent of coronary disease. Therefore, QTDex reflects a dimension of scintigraphic ischemia unrelated to the extent of angiographic coronary disease.

11:30

880-5 Effects of Exercise Training in Patients With Acute Myocardial Infarction Treated With Primary Percutaneous Transluminal Coronary Angioplasty

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Background: Exercise training after acute myocardial infarction (AMI) improves exercise capacity but data for patients treated with primary percutaneous transluminal coronary angioplasty (PTCA) are scarse. It is also less clear whether patients with large infarctions should be included in training programs, since some studies suggest that unfavorable left ventricular remodeling can occur during training.

Methods: We evaluated the training effect (change in VO2 max and total Watts performed at bicycle ergometry after 6 weeks training) in 20 consecutive patients (mean age 51 years sd 13, 18 men) with a first AMI succesfully treated with primary PTCA. Infarct size was determined before and after training, using quantitative (PERFITT program, Nuclear Diagnostics) stress-rest 201-Thallium single-photon emission computed tomography. Two subgroups were identified: group I (N = 11) with normal radionuclide ejection fraction (EF) at discharge and group II (N = 9) with reduced EF (59% sd 3 in group I versus 44% sd 13 in group II, $p < 0.0001$).

Results: Training results are summarised in the table (data expressed as median, non parametrical statistics for differences before and after training).

	Before training	After training
Total group (N = 20)		
Total Watts	1000	1175 (**) = +18%
VO2 max (ml/kg/mn)	19	22 (**) = +23%
Thallium defect size (%)	4	6 (ns)
Group I (N = 11)		
Total Watts	1000	1175 (*) = +18%
VO2 max (ml/kg/mn)	19	22 (*) = +16%
Thallium defect size (%)	3	3 (ns)
Group II (N = 9)		
Total Watts	850	1175 (**) = +38%
VO2 max (ml/kg/mn)	18	22 (*) = +22%
Thallium defect size (%)	18	12 (p = 0.13)

(*): $p < 0.05$, (**) $p < 0.01$ and ns: not significant

Conclusions: In patients with a first AMI treated with primary PTCA, exercise capacity improves after physical training. This benefit is seen in patients with normal and reduced EF and is not associated with an increase in infarct size. There is even a trend toward a decrease in infarct size in patients with reduced EF.

11:45

880-6 Retrospective Automated Analysis of Risk After Exercise Testing and Appropriate Therapy

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Quality control is best tested when required parameters are extracted from routinely recorded clinical procedures. Since 1987 exercise testing and cardiac catheterizations have been recorded electronically in a VA centralized hospital database (DHCP). The Mark score for exercise testing, reported in N Engl J Med, uses peak ST deviation, angina index, and exercise duration to categorize patients into low (<1% mortality/year), medium (2–4%), and high risk (>4%/year). Risk estimates can be adequately approximated by the formula $Pr(X) = (0.0157 + 0.0008X)/(1 + 0.0399X)$, where X = duration − 5 ST − 4 × angina index. Some 6,574 VA patients' exercise tests were evaluated, using Mark's publication date (9/91) to group results.

Mortality Risk	Total	Before publication	After publication
Low	5465 (83.1%)	2652 (82.8%)	2813 (83.4%)
Med	896 (13.6%)	461 (14.4%)	435 (12.9%)
Hi	213 (3.3%)	88 (2.7%)	125 (3.7%)

Since catheterization is appropriately recommended for those at high risk, the database was further searched for high risk patients who did not receive catheterization. Some 33 patients with a high risk exercise test did not undergo catheterization (15.5%) (13 before publication and 20 after publication, p = NS). High risk during exercise testing can be computed retrospectively to reveal patients improperly treated. Performance of this measure was not better before the Mark et al. classification publication than after.

881 In-Stent Restenosis Mechanisms

Wednesday, April 1, 1998, 10:30 a.m.–Noon
Georgia World Congress Center, Lecture Hall 2

10:30

881-1 Early and Late Vascular Responses to Coronary Stenting in Humans

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Background: Despite widespread use of coronary stents, only limited pathologic data following placement in humans are available.

Methods: Histology on 50 stents in 30 coronary arteries from 28 patients (mean age 64 ± 10 years, 16 men, 12 women) was performed.

Results: The clinical diagnosis at the time of stenting was unstable angina in 11 cases, acute MI in 7, and stable angina in 10. The mean duration of stent placement was 32 ± 78 days (range 0.5–390 days). Fibrin, platelets, and PMN's were associated with cement wires in stents examined ≤11 days after implantation. When present, a lipid core (LC) was focally penetrated by stent wires in 27% of arterial segments. 442 stent wire sites from stents ≤3 days after implant were analyzed, and inflammation associated with stents was related to the underlying plaque and artery wall: 97% of struts in contact with fibrous plaque (FP) had ≤20 associated inflammatory cells compared with 56% of struts embedded in LC and 64% of struts in contact with damaged media. In contrast, only 3% of struts in contact with FP had >20 associated inflammatory cells compared with 44% of struts embedded in LC and 36% of struts in contact with damaged media ($p < 0.001$). Neointimal cells/mm² in arteries stented ≥70 days was 3280 ± 869 and was similar to PTCA artenes (3260 ± 851 cells/mm²) matched for time since treatment (195 ± 131 days for stents and 180 ± 137 days for PTCA). Alcian blue staining of stents and matched PTCA arteries showed similar patterns of proteoglycan (hyaluronic acid, chondroitin/dermatan, and heparan suffate) deposition.

Conclusions: The vascular responses to stenting provide targets for their improved outcome via reduction in early thrombus formation, attenuation of inflammation, and avoidance of acute medial injury. Similar neointimal cell density and proteoglycans in stents and PTCA suggest common approaches to treat restenosis.

10:45

881-2 Histologic Responses During In-stent Neointimal Regression in a Porcine Coronary Model

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Backgound: Clinically, in-stent neointima has been reported to regress over time, the mechanism of which is unknown.

Methods: To assess the phenomenon of in-stent neointima regression, NIR™ stents were implanted in porcine coronary artenes (stent/artery = 1.1) and harvested at 2 and 6 mos (N = 4 stents/group). Histopathologic analysis included morphometric analysis (Movat stain), smooth muscle cell (SMC) density/hpf (H&E), collagen content (Sirus red stain), and proteoglycan content (Alcian blue stain ± hyaluronidase = hyaluronic acid).

Results: There was a significant reduction in neointima at 6 mos despite similar injury scores (0.05 ± 0.06 vs 0.36 ± 0.29 at 6 mos).

	Area	Thickness	Stenosis
2 months	1.4 ± 0.4 mm²	0.2 ± 0.03 mm	21 ± 4%
6 months	0.8 ± 0.1 mm²*	0.02 ± 0.01 mm*	14 ± 2%*

*P < 0.05 vs 2 mos.